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Thunderstorm associated asthma: a detailed analysis of environmental factors

Antonio Celenza, Jane Fothergill, Emil Kupek, Rory J Shaw

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St Mary's Hospital, London W2 1NY
Antonio Celenza, *registrar in accident and emergency medicine*
Jane Fothergill, *consultant in accident and emergency medicine*
Emil Kupek, *research assistant in statistics*
Rory J Shaw, *consultant respiratory physician*

Correspondence to: Dr Fothergill.

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Abstract

***Objectives*—To seek associations between meteorological factors, concentrations of air pollutants or pollen, and an asthma epidemic which occurred in London on 24 and 25 June 1994 after a thunderstorm.**

***Design*—Retrospective study of patients' accident and emergency department records, with bivariate and multivariate analysis of environmental factors and data collection for the two months surrounding the epidemic.**

***Setting*—The accident and emergency department of St Mary's Hospital in west central London.**

***Subjects*—148 patients presenting with asthma between 1 June and 31 July 1994, of whom 40 presented in the 24 hours after the storm.**

***Results*—The asthma epidemic was significantly associated with a drop in air temperature six hours previously and a high grass pollen concentration nine hours previously. Non-epidemic asthma was significantly associated with lightning strikes, increase in humidity or sulphur dioxide concentration, a drop in temperature or high rainfall the previous day, and a decrease in maximum air pressure or changes in grass pollen counts over the previous two days.**

***Conclusions*—New episodes of asthma during the epidemic on 24 and 25 June 1994 were associated with a fall in air temperature and a rise in grass pollen concentration. Non-epidemic asthma was significantly associated with a greater number of environmental changes. This may indicate that the patients with thunderstorm associated asthma were a separate population, sensitive to different environmental stimuli.**

Introduction

On 24 and 25 June 1994 an acute outbreak of asthma occurred in southern England associated with a thunderstorm.[1 2] The abrupt rise in the number of patients presenting with asthma to the accident and emergency department of St Mary's Hospital in west central London provided an opportunity to assess whether there had been any precipitating environmental factors. Data on meteorological changes and local concentrations of air pollutants and grass pollen were collated for the two months surrounding the epidemic to see whether these factors were temporally associated with cases of non-epidemic asthma presenting during the study period or with the asthma epidemic itself.

Patients and methods

We analysed the records of patients who had presented with asthma to the accident and emergency department between 1 June and 31 July 1994. Patients aged 16 or over who were assessed by a doctor and given a diagnosis of asthma were included. Patients who refused proper assessment or in whom the diagnosis was unclear were excluded. Only one patient reattended with asthma during the study period, and this was before the epidemic.

Three hourly measurements of rainfall, ambient temperature, barometric pressure, and humidity were obtained from the Meteorological Office. Measurements were taken at the London Weather Centre, Holborn, roughly 4 km from St Mary's Hospital. Hourly measurements of ground lightning strikes were obtained from EA Technologies, Capenhurst, Chester, which used vector analysis of current detected by local surface electrodes. Vector analysis covered a 10 km radius centred on St Mary's Hospital. Daily hourly maximum concentrations of the air pollutants nitrogen dioxide, sulphur dioxide, and ozone were obtained from Westminster Council. Hourly measurements of these variables were obtained for 24 and 25 June. Measurements were taken from Marylebone Road, roughly 1·5 km from St Mary's Hospital. Daily grass pollen counts with two hourly measurements for 24 and 25 June were obtained from the Pollen Research Unit, University of North London. Measurements were taken from the University of North London building in Holloway, roughly 6 km from St Mary's Hospital. Fungal spore concentrations were not available.

STATISTICAL ANALYSIS

Over the two month study period daily measurements were obtained either as totals for the 24 hours (asthma presentations, lightning strikes, rainfall), as maximum values for the 24 hours (air pollutant and grass pollen concentrations), or as the maximum changes that occurred during any three hour period during the day (temperature, pressure, humidity). More detailed three hourly environmental data were obtained for the period surrounding the thunderstorm and epidemic on 24 and 25 June. Cut points for the independent variables were selected so that the baseline values represented typical summer weather conditions.

The two datasets were analysed separately. Poisson regression was used to relate the number of reported asthma cases and environmental variables, including lagged effects over 24 or 48 hours for the daily data and lagged effects over three, six, or nine hours for the three hourly data. The independent variables were first fitted in bivariate analysis to see which individual environmental changes seemed to be associated with increased presentations with asthma. The final multivariate models were arrived at by systematically eliminating the variables least significantly associated with the outcome and then refitting the models. Multivariate models thus took into account several interrelated environmental factors and showed which were most strongly associated with asthma presentations. Analysis was carried out with SAS software to find the best predictors of episodes of asthma.[3]

Owing to lagging of possible cause and effect and missing values, two out of 17 and eight out of 61 datapoints were lost for the multivariate analysis of hourly data and daily data respectively. The missing values call for caution when interpreting the results, especially for the lagged models.

Results

The significant associations with presentations of asthma calculated with the multivariate model are shown in table 1. The "parameter estimate" was derived to show the relative change in asthma presentations compared with those during baseline environmental conditions. A parameter estimate of 1·00 indicates presentations during baseline summer environmental conditions in London, higher figures indicating the factor by which presentations rose. The table lists both daily variables for total asthma cases and three hourly data for epidemic asthma. Figures 1 and 2 show those variables with significant associations with asthma presentations in a multivariate Poisson regression model.

In an attempt to define to what extent the multivariate model explained the variation in asthma presentations we calculated the percentage deviance from the null hypothesis. The multivariate model for daily data during the two months surrounding the epidemic reduced the deviance from 167·69 to 36·23—a 78·4% reduction—indicating that the model identified most environmental factors associated with non-epidemic asthma presentations. Similarly, during the period of the epidemic the multivariate model for hourly data reduced the deviance by 84·1% (from 46·42 to 7·36), indicating that most environmental factors associated with the epidemic were identified. Owing to the storm some atmospheric variables were closely related, as indicated by their moderate to high correlation in the multivariate models (details not shown).

Table 1—*Environmental associations with asthma presentations (multivariate Poisson regression model)*

	Parameter estimate	Confidence interval Lower	Confidence interval Higher	P value
Three hourly data from asthma epidemic				
Meteorological data:				
Fall in air temperature three hours previously (°C)	1·19	1·05	1·35	0·01
Fall in air temperature six hours previously (°C)	1·09	1·01	1·19	0·04
Grass pollen:				
Pollen count >74 grains/l	0·18	0·07	0·49	<0·01
Pollen count 29-74 grains/l	0·26	0·12	0·56	<0·01
Pollen count 1-28 grains/l	0·29	0·09	0·98	0·05
Pollen count zero	1·00†			
Fall in pollen count by >30 grains/l from nine hours before	2·50	1·36	4·59	<0·01
Rise in pollen count by >30 grains/l from nine hours before	0·83	0·42	1·67	0·61
Missing values	0·40	0·12	1·32	0·13
Change in pollen count by <30 grains/l over previous nine hours	1·00†			
Daily data for June and July 1994				
Meteorological data:				
>10 Lightning strikes	2·57	0·84	7·84	0·10
1-9 Lightning strikes	3·21	1·65	6·23	<0·01
No lightning strikes	1·00†			
Rise in maximum humidity (%)	1·04	1·01	1·07	0·02
Fall in maximum air temperature previous day (°C)	1·11	1·05	1·18	<0·01
Less rainfall than previous day	2·70	1·60	4·56	<0·01
More rainfall than previous day	0·36	0·13	0·97	0·04
Same rainfall as previous day	1·00†			
Fall in maximum air pressure two days before (hPa)	1·06	1·03	1·10	<0·01
Air pollutants:				
Rise in sulphur dioxide concentration by >20 ppm from previous day	2·07	1·35	3·17	<0·01
Fall in sulphur dioxide concentration by >20 ppm from previous day	0·95	0·54	1·66	0·85
Change in sulphur dioxide concentration by <20 ppm from previous day	1·00†			
Grass pollen:				
Fall in pollen count by >10 grains/l from two days before	1·89	1·24	2·89	<0·01
Rise in pollen count by >10 grains/l from two days before	2·15	1·21	3·84	0·01
Change in pollen count of <10 grains/l from two days before	1·00†			

†Baseline.

EPIDEMIC ASTHMA

Meteorological factors

During the two month study period (1 June to 31 July) 148 patients who presented with asthma were included. A mean of 2·25 patients with asthma present to the accident and emergency department daily; but in the 24 hours from 2100 on 24 June, 40 patients presented. During the three hours before then—that is, between 1800 and 2100 on the 24th—there was a thunderstorm over London. During the storm there were 50 groundstrikes of lightning, a rise in humidity from 50% to 87%, a rainfall of 46 mm, and a rise in the grass pollen count from 37 to 130 grains/l. In the three hours after the storm—that is, from 2100 to 2400—the air temperature fell from 26·5°C to 18·7°C.

Bivariate analysis of the three hourly data showed that the number of lightning strikes, increase in rainfall, fall in temperature, and rise in air pressure and relative humidity were each associated with significant increases in epidemic asthma presentations six hours later. Decrease in air temperature and a rise in air pressure were also significantly related to asthma presentations with a lag of three hours. Once the asthma epidemic was under way the associated environmental factors were a fall in air temperature and air pressure and an increase in relative humidity. Air pressure rose before the thunderstorm, then fell sharply. Lag periods of three to six hours in asthma presentations may represent the time taken for patients to become unwell and present to hospital. A drop in air temperature six hours previously was associated with an increase in asthma presentations by a factor of 1·09 per degree centigrade fall ($P<0·05$) in the multivariate model.

Grass pollen

Changes in grass pollen concentration were also associated with increases in asthma presentations in the multivariate model (see table). There was a peak in pollen concentration roughly nine hours before the peak in asthma presentations (fig 2).

Air pollutants

When the acute epidemic was considered there was no evidence that concentrations of vehicle exhaust pollutants were independently related to the increase in asthma presentations.

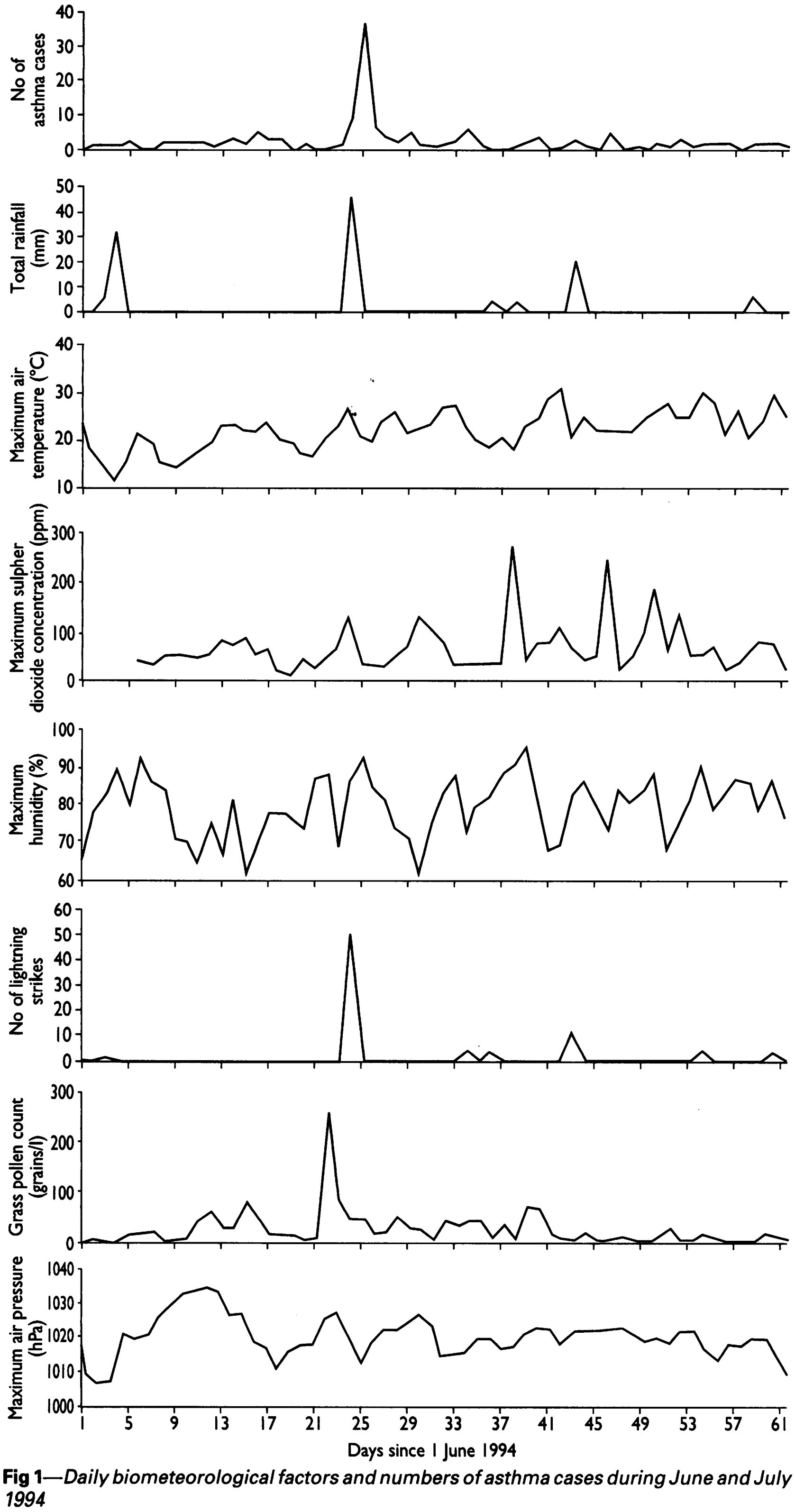

Fig 1—*Daily biometeorological factors and numbers of asthma cases during June and July 1994*

NON-EPIDEMIC ASTHMA

When the whole two month study period was considered, meteorological factors, air pollutants, and grass pollen concentration were all important predictors of presentations with asthma. However, daily data for this period included both epidemic and non-epidemic asthma. Multivariate analysis partially controlled for this by using the difference in number of cases of asthma between June and July.

Meteorological factors

Both bivariate and multivariate analysis showed a significant association between lightning strikes and asthma presentations during June and July ($P<0{\cdot}05$). Multivariate analysis showed that a figure of between one and nine lightning strikes a day was associated with an increase in asthma presentations by a factor of 2·21 compared with when there was no lightning ($P<0{\cdot}05$). Bivariate and multivariate analysis of the other meteorological variables suggested that there were significant associations of increased cases of non-epidemic asthma with high rainfall or a drop in maximum air temperature the previous day and a fall in air pressure or an increase in relative humidity the same day or up to two days previously.

Air pollutants

The bivariate model showed that relatively low sulphur dioxide and nitrogen dioxide concentrations were significantly associated with a decrease in the number of patients presenting with asthma. Multivariate analysis suggested that a rise in sulphur dioxide concentration the previous day was the only measure of air pollution which was associated with presentations of asthma. A rise in sulphur dioxide concentration by more than 20 ppm compared with the previous day was associated with an increase in asthma presentations by a factor of 2·07 ($P<0.01$). No association was found between ozone concentrations and asthma presentations.

Grass pollen

As expected, change in grass pollen concentration was also significantly associated with asthma presentations after a lag of 48 hours. This association was with both a rise and a fall in grass pollen concentrations, indicating the importance of lag effects. This may also reflect the mixture of epidemic and non-epidemic asthma populations in the daily data, who may respond differently and at different times to a grass pollen stimulus.

Discussion

EPIDEMIC ASTHMA

Published reports of asthma epidemics around the world have related asthma presentations to various environmental factors, such as atmospheric temperature inversions,[4] fog,[5] and organic dusts.[6 7] Thunderstorms in which fungal spores and rye grass pollen were implicated as the precipitating factors in

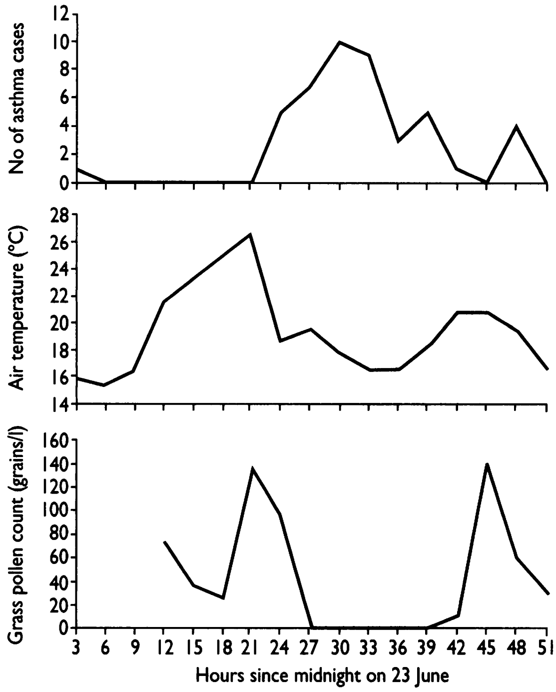

Fig 2—*Predictors of numbers of asthma cases during epidemic (multivariate model)*

Key messages

- A thunderstorm in London during the evening of 24 June 1994 was followed by an asthma epidemic. The epidemic was significantly associated with a fall in air temperature six hours before the storm and a high grass pollen count nine hours before
- Non-epidemic asthma during June and July 1994 was significantly associated with lightning strikes, increase in humidity or sulphur dioxide concentration, a fall in temperature or high rainfall the previous day, and a decrease in maximum air pressure or changes in grass pollen concentration over the previous two days
- Patients with asthma associated with thunderstorms may be a separate population from other patients with asthma, sensitive to different environmental stimuli

asthma epidemics occurred in Birmingham in 1983[8 9] and Melbourne in 1984.[10-12] Campbell-Hewson *et al* reported that the asthma epidemic in Peterborough and Cambridge in June 1994 was associated with a thunderstorm in which there were high ozone concentrations, high pollen counts, and high fungal spore counts with varying levels of atmospheric electrical activity.[2] Our findings in west central London suggest that a fall in air temperature and a high pollen count were the best predictors of the asthma epidemic, though all the individual environmental variables were closely related.

The sudden thunderstorm caused the greatest and most sudden fall in temperature and air pressure as well as the most pronounced increase in humidity, rainfall, and lightning strikes observed during the two month study period. As shown in figure 1, lightning strikes, rainfall, air temperature, air pressure, and humidity seemed to be related to the onset of the asthma epidemic. We could not identify from the data which of the environmental factors caused the epidemic of asthma, as similar but more modest changes in environmental conditions occurred on other days during the study period without increases in asthma presentations.

The meteorological change which showed the best association with a rise in new asthma cases was a fall in air temperature six hours previously. We did not look for temperature inversions. Inversions may reduce the upward dispersion of pollutants and allergens, leading to their accumulation near the ground. Though temperature inversions have been implicated in triggering asthma by other studies, it is unlikely that an inversion occurred on 24 June, as air pollutant concentrations were not unduly high.

The changes in meteorological conditions were accompanied by a sharp rise in grass pollen concentration shortly after the thunderstorm. Statistical analysis of grass pollen concentration showed a significant relation with asthma presentations, with a lag of nine hours. This supports findings of other studies suggesting that release of inhalable grass pollen after rainfall may be associated with an increase in asthma presentations.[11 12] Sensitivity to grass pollen was not assessed in patients presenting with asthma during the study period.

It seems unlikely that high concentrations of vehicle exhaust pollutants were the precipitating factor for the asthma epidemic. High concentrations of ozone or nitrogen dioxide have been shown to increase bronchial responsiveness in other studies.[13-15] However, concentrations of these pollutants were not exceptionally high in the days preceding or during the asthma epidemic. Our data differ from those from Cambridge, a city in a more rural location, in which there was a comparatively high concentration of ozone during the epidemic.[2]

NON-EPIDEMIC ASTHMA

Attendances for asthma have consistently been significantly associated with air pollution[13-17] but results of studies outside epidemics in relation to meteorological factors have been contradictory. Rainfall,[15] high humidity,[18] low wind speed,[15 18] high air pressure and high temperature,[18] as well as low temperature,[19] have all been associated with asthma presentations. This study suggests associations between asthma presentations in June and July 1994 and complex, interrelated combinations of meteorological factors, air pollutants, and grass pollen as noted in the results of the bivariate analysis. Multivariate analysis suggested that lightning strikes, rise in humidity or in sulphur dioxide concentrations, a fall in temperature or relatively high rainfall the previous day, and a fall in air pressure or changes in grass pollen concentration over the previous two days were the best independent predictors of non-epidemic asthma presentations.

In conclusion, the thunderstorm associated sudden fall in air temperature and sudden rise in grass pollen concentration were independently associated with a rise in epidemic asthma presentations to our accident and emergency department. Several environmental changes which occurred immediately before and during the thunderstorm were also significantly temporally associated with the epidemic. This supports previous findings that under certain conditions thunderstorms may precipitate an asthma epidemic. Our study also suggests that the factors associated with non-epidemic asthma differed from those associated with the epidemic. This indicates that the many patients attending the accident and emergency department with reversible airways obstruction related to the thunderstorm may have been a different population, sensitive to different environmental stimuli.

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Conflict of interest: None.

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